# CXR #9

WAMSS SGR 2022

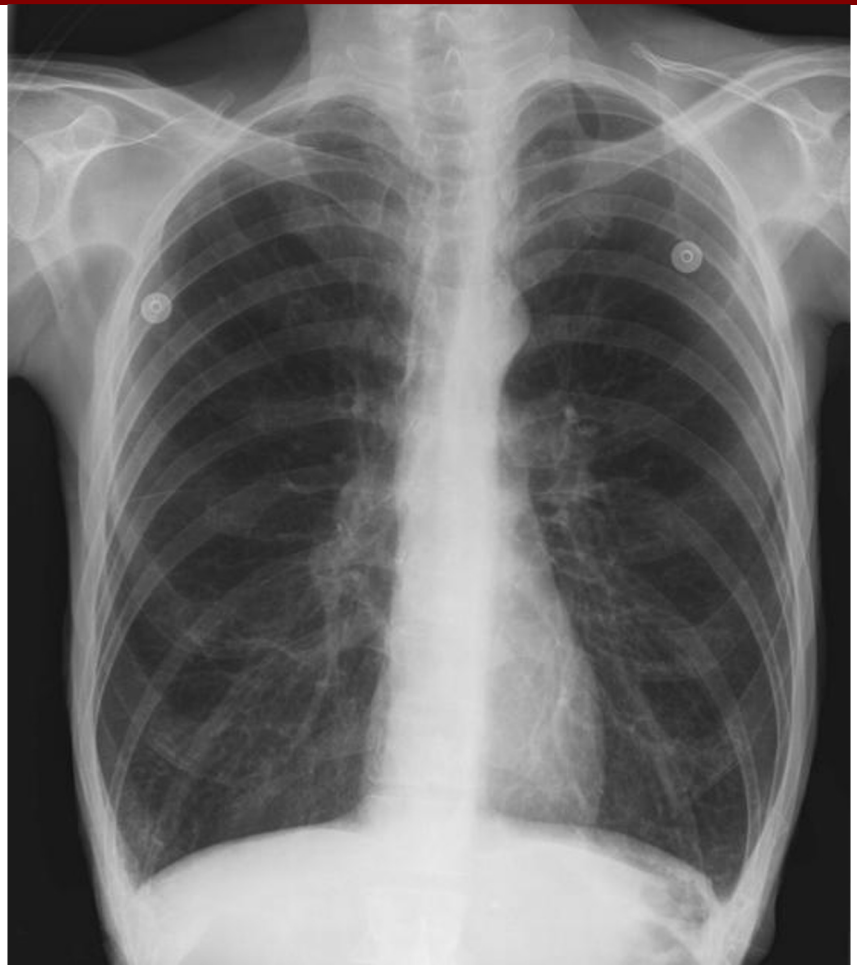
# Trigger

You are a GP working in Peppermint Grove. Kaiba, a 69M with a 51 pack year smoking history presents with progressive dyspnoea over the past year. He can walk 5m before onset of dyspnoea and chest tightness. Kaiba previously used to go on daily 2km walks with his wife and daughter. His wife was helping with his ADLs. He has gained 5kg over the past year and now has a BMI of 31.

Kaiba has a past history of Hepatitis C acquired from IVDU which has been cleared and has had a prostatectomy to treat prostate cancer 5 years prior. He has no other significant medical history.

A PA CXR was performed on admission.

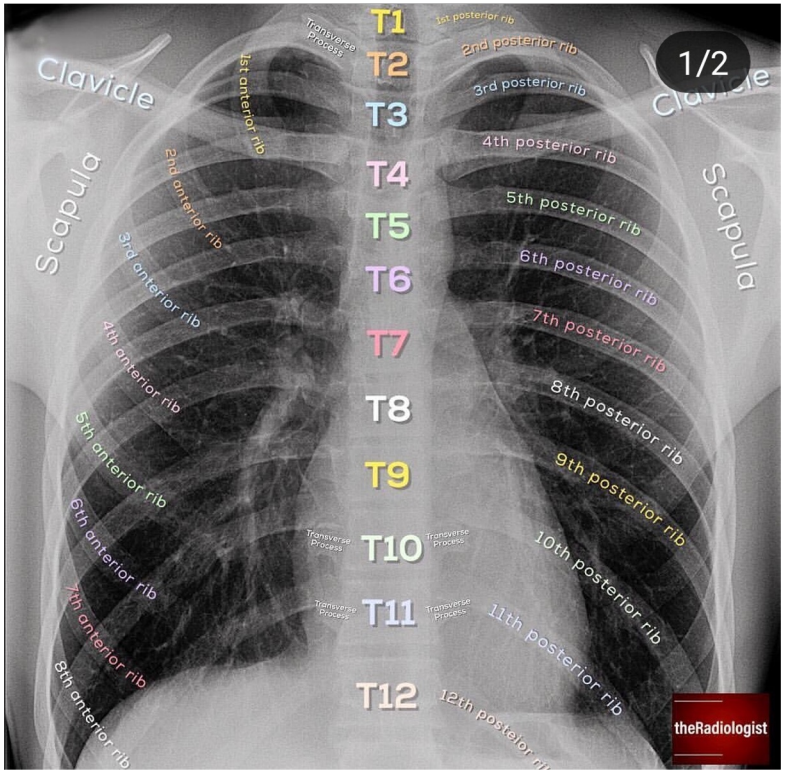
**Task**: Interpret the PA CXR and provide a working diagnosis.



| | |
|---|---|
| **Details and demographic** | PA CXR of a 69M with a 51 pack year smoking history presenting with progressive dyspnoea for over a year |
| **RIPE/Quality** | Rotation : Slight rotational artefact to the right (distance between clavicle and spinous process is wider on the right) |
| | Inspiration: Adequate inspiratory effort |
| | Projection: PA |
| | Exposure: Adequate exposure |
| **Airways and lung fields** | Trachea is equidistant between the two clavicles, suggesting no tracheal deviation<br>Bilateral diffuse hyperlucency<br>No abnormal opacifications |
| **Bones and soft tissue** | No obvious fractures or soft tissue abnormalities<br>6 anterior and 10 posterior ribs visible (normal finding) |
| **Cardo-mediastinum** | Cardiothoracic ratio is low (<0.42) |
| **Diaphragm** | Flattened hemidiaphragms with widened costophrenic angles |
| **Everything else** | No free gas under the diaphragm<br>2 ECG electrodes present |
| **Interpretation** | PA CXR of a 69M with a 51 pack year smoking history presenting with progressive dyspnoea for over a year.<br>The lungs are hyperlucent bilaterally and the hemidiaphragms are flattened with widened costophrenic angles. Additionally, the cardiothoracic ratio is low. This is suggestive of **lung hyperexpansion**. Given the patient's 51 pack year history, my working differential is **Chronic Obstructive Pulmonary Disease (COPD)**. |

# Counting Ribs

- >6 anterior ribs can be a sign of hyperexpansion
- >10 posterior ribs can be a sign of hyperexpansion
- Don't forget to count the posterior rib at the top!
- Hyperexpansion -> barrel chest



# Follow-up Questions

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1. What are the spirometry findings of COPD?

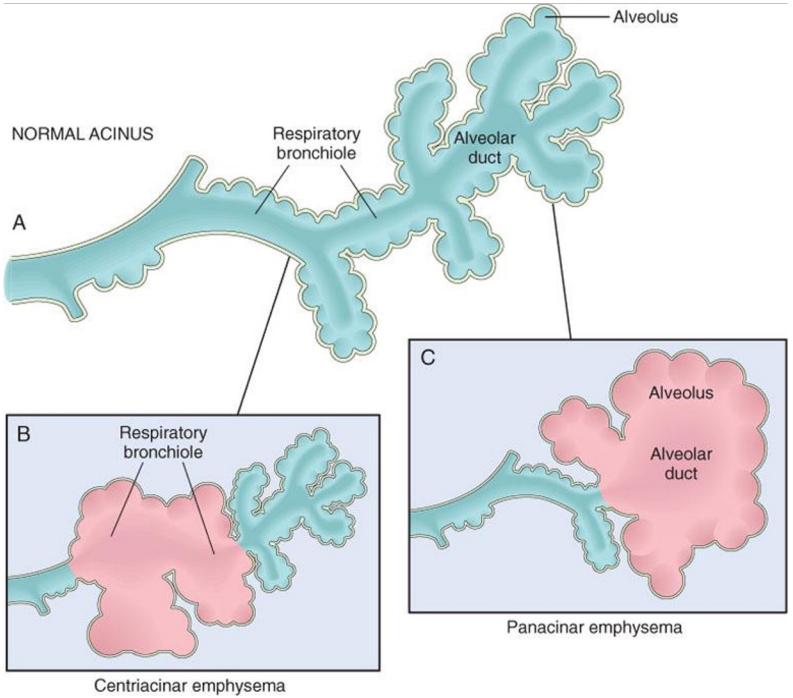
2. What are the 2 morphological subtypes of emphysema and what are their most common aetiologies?

# Question 1

- FEV1/FVC < 70%
- In obstructive lung diseases, FEV1 decreases and FVC decreases by less or is normal, so FEV1/FVC is low
- TLC increases as a result of air trapping due to obstruction (but may be normal)
- <12% improvement in FEV1/FVC post-bronchodilator
    - Reversible airflow obstruction is associated with asthma, not COPD

# Question 2

- Centriacinar emphysema
  - Classically seen in smokers
  - Destruction of respiratory bronchioles that spares distal alveoli
  - Usually affects upper lobes
- Panacinar emphysema
  - Associated with α1-antitrypsin deficiency
  - Destruction of entire acinus (respiratory bronchiole + alveoli)
  - Usually affects lower lobes
- Mnemonic: smoke doesn't reach the distal alveoli in centriacinar emphysema, whereas with α1-antitrypsin deficiency it affects the whole bronchiole. Smoke 'rises up' to affect the upper lobes in centriacinar emphysema

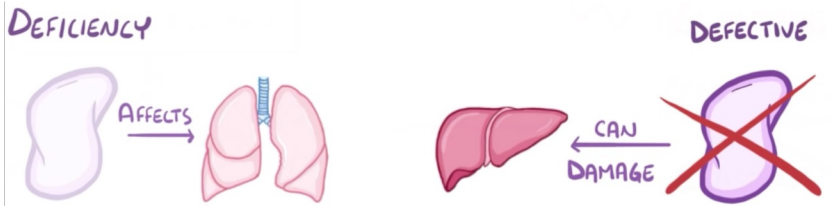


1. What are the spirometry findings of COPD?

2. What are the 2 morphological subtypes of emphysema and what are their most common aetiologies?

3. Outline the pathophysiology of α1-antitrypsin deficiency and describe its clinical presentation.

# Question 3: Pathophysiology




- α1-antitrypsin is a protease inhibitor produced in the liver. An autosomal recessive genetic mutation leads to α1-antitrypsin being misfolded.
- When misfolded α1-antitrypsin builds up in the liver (specifically the endoplasmic reticulum) it causes hepatocyte death.
    - This leads to hepatitis and cirrhosis
- Neutrophil elastase breaks down proteins. It's normal function is to break down bacteria in the lungs. α1-antitrypsin inhibits neutrophil elastase.
- A deficiency in α1-antitrypsin leads to uninhibited neutrophil elastase activity, breaking down elastin in the lungs (not just bacteria anymore), leading to lung parenchyma destruction.
    - This leads to panacinar emphysema

# Question 3: Clinical Presentation

- Pulmonary manifestations
  - Cough
  - Wheezing
  - Dyspnoea
  - Diminished breath sounds
  - Barrel chest
- Hepatic manifestations: history
  - Fatigue/malaise/weight loss
  - Pruritus
  - Bruising
  - Hypogonadism e.g. libido loss, erectile dysfunction
  - Decreased body hair
- Hepatic manifestations: examination
  - Weight loss
  - Jaundice
  - Spider naevi
  - Caput medusae
  - Palmar erythema
  - Bruising
  - Nail clubbing
  - Terry's nails
  - Hepatomegaly
  - Splenomegaly
  - Ascites
  - Gynaecomastia
  - Decreased body hair

# Thank you!

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